Thermoregulation during cold water immersion is unimpaired by low muscle glycogen levels

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YOUNG, ANDREW J., MICHAEL N. SAWKA, P. DARRELL NEUFER, STEPHEN R. MUZA, ELDON W. ASKEW, AND KENT B. PANDOLF. *Thermoregulation during cold water immersion is unimpaired by low muscle glycogen levels.* J. Appl. Physiol. 66(4): 1809–1816, 1989.—This investigation studied the importance of muscle glycogen levels for body temperature regulation during cold stress. Physiological responses of eight euglycemic males were measured while they rested in cold (18°C, stirred) water on two separate occasions. The trials followed a 3-day program of diet and exercise manipulation designed to produce either high (HMG) or low (LMG) preimmersion glycogen levels in the muscles of the legs, arms, and upper torso. Preimmersion vastus lateralis muscle glycogen concentrations were lower during the LMG trial (144 ± 14 mmol glucose/kg dry tissue) than the HMG trial (543 ± 53 mmol glucose/kg dry tissue). There were no significant differences between the two trials in shivering as reflected by aerobic metabolic rate or in the amount of body cooling as reflected by changes in rectal temperature during the immersions. Postimmersion muscle glycogen levels remained unchanged from preimmersion levels in both trials. Small but significant increases in plasma glucose and lactate concentration occurred during both immersions. Plasma glycerol increased during immersion in the LMG trial but not in the HMG trial. Plasma free fatty acid concentration increased during both immersion trials, but the change was apparent sooner in the LMG immersion. It was concluded that thermoregulatory responses of moderately lean and fatter individuals exposed to cold stress were not impaired by a substantial reduction in the muscle glycogen levels of several major skeletal muscle groups. Furthermore, the data suggest that, depending on the intensity of shivering, other metabolic substrates are available to enable muscle glycogen to be spared.

shivering; thermogenesis; body temperature regulation

HUMANS HAVE TWO PRIMARY physiological mechanisms available to maintain normal body temperature during cold exposure. Cutaneous vasoconstriction increases tissue insulation and limits convective and conductive heat transfer from the body to the environment. Except for very mild cold stress conditions, however, cutaneous vasoconstriction alone is insufficient to prevent a loss of body heat stores, and an increase in metabolic heat production is necessary to prevent body temperature from decreasing. In the absence of a voluntary increase in muscular activity, metabolic heat production will be increased during cold exposure by involuntary shivering. Like all types of muscular activity, shivering thermogenesis is dependent on an adequate supply of metabolic energy substrates. Although the principal substrate has not been clearly identified, both blood glucose and muscle glycogen have been reported to be metabolized during shivering thermogenesis.

There is ample evidence that plasma glucose plays some role in shivering thermogenesis. Gale et al. (12) found that when insulin was infused into subjects exposed to cold air, shivering stopped as plasma glucose concentration fell below 2.5 mmol/l; with the cessation of shivering, metabolic rate, and core temperature decreased. Intravenous glucose administration rapidly restored shivering, but, even more interestingly, shivering was restored in both an arterially occluded leg as well as a nonoccluded leg, suggesting that the glucose effect on shivering was via the central nervous system as opposed to a peripheral mechanism (12). Haight and Keatinge (14) exposed subjects to cold air after a 2-h bout of heavy exercise followed by ethanol ingestion. This manipulation was employed to produce hypoglycemia and resulted in less visible shivering, lower metabolic rates, and greater body temperature declines compared with responses observed when glucose ingestion followed the ethanol and prevented hypoglycemia.

Whether muscle glycogen is a substrate for shivering thermogenesis is not clear. Muscle glycogen utilization was not assessed during the experiments of Haight and Keatinge (14) mentioned above; however, the heavy exercise bouts undoubtedly produced, in addition to hypoglycemia, some degree of glycogen depletion in several large muscle groups. Thus, glycogen depletion may have, at least partially, accounted for the effects attributed to hypoglycemia. Animal experiments suggest that during shivering there is a decrease in plasma glucose oxidation and a concomitant increase in muscle glycogenolysis compared with rest (21). Other animal experiments suggest that the improvement in cold tolerance produced by cold adaptation may be related to a concomitant increase in liver and skeletal muscle glycogen levels (18). In addition, muscle glycogen utilization in humans performing low-intensity exercise was observed to be greater in a cold compared with temperate environment, and the additional glycogen utilization was attributed to the energy cost of shivering (16). Thus, the relative importance of these different metabolic substrates for satisfying the energy requirements of shivering thermogenesis remains unclear. Despite the lack of experimental evidence, one widely cited mathematical model of human thermoregu-

lation bases its prediction of tolerance time in cold water on the rate of glycogen depletion in the skeletal muscle (26).

The purpose of the present investigation was to study the importance of muscle glycogen for body temperature regulation during acute cold stress. Specifically, the experimental aim was to measure thermoregulatory and metabolic responses during cold water immersion in resting euglycemic humans with high vs. low muscle glycogen levels. It was hypothesized that the shivering and metabolic heat production would be impaired and body cooling would be greater when subjects were immersed in cold water in a glycogen-depleted state. In addition, the experimental design offered an opportunity to determine whether or not a prolonged period of shivering during cold water immersion would result in a significant depletion of muscle glycogen stores in otherwise resting individuals.

METHODS

Subjects and experimental design. Eight male volunteers served as test subjects in this investigation after being completely informed as to the risks and requirements of participation. Before experimental testing, each subject's maximal O_2 uptake ($\dot{V}O_{2max}$) was determined and anthropometric measures were assessed.

Each subject completed two experimental cold water immersion tests. The immersions were separated by at least 14 days to control for possible effects of repeated cold water immersion (27). One cold water immersion was completed with the subjects having high muscle glycogen (HMG) levels and the other with low muscle glycogen levels (LMG). The subjects completed the trials in a randomized order, with three subjects undergoing the HMG trial before the LMG trial. The cold water immersions were performed on the same day of the week and at the same (within 1 h) time of day. A fasting period of at least 10 h preceded the immersions, during which the subjects were permitted to ingest only water.

The cold water immersion tests were performed in a 36,000-liter pool. The water was continuously circulated, and the temperature was maintained at 18°C for all immersions. The subjects wore swimming trunks for the immersion tests. Approximately 1 h before the scheduled immersion time, subjects were weighed and instrumented and had a catheter placed in the antecubital vein of one arm. A muscle sample was obtained from the vastus lateralis ~30-min before immersion, after which the subjects moved to a nylon-mesh lounge chair where they reclined in a semisupine position while wrapped in blankets. After 20-min of quiet rest in this position, a blood sample was obtained, and base-line measurements of rectal temperature (T_{re}) and respiratory exchange parameters were completed. The platform supporting the chair was then lowered into the pool, and the water level was adjusted to the base of the subjects' neck. The subjects rested quietly throughout the immersion, which was terminated after 180 min or when T_{re} fell below 35.5°C. During immersion, T_{re} and skin heat flow (h_c) were continuously measured, and respiratory exchange parameters were measured at 5-min intervals. The electrocardiogram was continuously monitored and heart rate was recorded at 10-min intervals. Blood samples were obtained at the 10th and 60th min of immersion. Immediately before removing the subjects from the water a final blood sample was obtained. On egressing from the water, the subjects were rapidly dried and wrapped in blankets. A final muscle sample was obtained from the vastus lateralis muscle within 5 min after completion of the immersion.

During the 3 days preceding the cold water immersion tests, the subjects resided in a dormitory where their diet and physical activity were strictly controlled to achieve the desired muscle glycogen levels. Before the LMG trial, a low-carbohydrate (15%), high-fat (65%) diet was consumed (2,900 kcal/day). In addition, the subjects completed an exercise regimen each day that consisted of three 60-min bouts of exercise at intensities sufficient to elicit 75% of their maximum heart rate. The three exercise bouts each employed a different mode of exercise (treadmill running, cycle ergometry, rowing ergometry), to deplete glycogen from several different major muscle groups of the arms, legs, and upper torso. During the 3 days preceding the HMG trial, a high-carbohydrate (65%), low-fat (15%) diet was consumed (3.200 kcal/day), and they were not permitted to exercise or engage in any strenuous activity. The menus for the two diets were designed by a dietitian to offer approximately equivalent total caloric content and were adjusted according to individual preference. All meals were prepared and served in measured portions under the supervision of the dietitian, and any food offered but not consumed was also measured, so that the individuals' actual intake could be determined. The subjects were instructed to consume only food prepared by the dietary staff during the 3 days preceding the cold water immersion tests. The diets and the procedures used to partition and quantify the subjects' food consumption have been described in detail elsewhere (5).

Experimental procedures. A continuous, progressive intensity treadmill test was used to determine $\dot{V}O_{2max}$ and the maximal heart rate (24). Measurements of $\dot{V}O_2$, carbon dioxide output ($\dot{V}CO_2$), and pulmonary minute ventilation ($\dot{V}E$) were made at 15-s intervals throughout this test using an automated system (Sensormedics Horizon MMC). Heart rates were determined from the electrocardiogram, which was monitored from chest electrodes (CM-5 placement) connected to an oscilloscope-cardiotachometer (Hewlett-Packard). Body density, determined by hydrostatic weighing, was corrected for residual lung volume and used to calculate percent body fat (10). Measurements of skinfold thickness at 13 sites distributed on the arm, leg, and torso were averaged to obtain mean skinfold thickness. Mean subcutaneous fat thickness was calculated by subtracting the thickness of two layers of skin (4 mm) from the mean skinfold thickness and then dividing by two (1).

During cold water immersion, $\dot{V}O_2$, $\dot{V}CO_2$, and $\dot{V}E$ were measured from the 6th through the 10th min of each consecutive 10-min period using the same automated system used in the maximal aerobic power test. Metabolic rate was calculated from the $\dot{V}O_2$ and the caloric

equivalent for O_2 appropriate for the measured respiratory exchange ratio (assumed equal to the nonprotein respiratory quotient). Heart rate was determined from electrocardiograms radiotelemetered to an oscilloscope-cardiotachometer. T_{re}, water temperature (T_w), and h_c were continuously measured and recorded throughout the experiment using a computer-operated system. A thermister placed ~50 cm from the subject was used to monitor T_w. A thermister inserted 10 cm beyond the anal sphincter was used to measure T_{re}. Measurements of h_c were obtained at five sites (upper chest, upper arm, left thigh, calf, and top of the foot) using h_c disks (RdF, Hudson, NH). The h_c disks were secured to the skin with one layer of double-backed adhesive tape. The disks were factory calibrated with a reported accuracy of 6–7% of actual h_c. Mean weighted heat flow ($\bar{h}_c$) was calculated according to the formula $\bar{h}_c = 0.47\ h_c$ (chest) + $0.14\ h_c$ (arm) + $0.19\ h_c$ (thigh) + $0.13\ h_c$ (calf) + $0.07\ h_c$ (foot), which is a slight modification of the equation of Hardy and DuBois (15). Tissue insulation was calculated as the difference between T_{re} and skin temperature (assumed to equal to T_w) divided by $\bar{h}_c$.

The pre- and postimmersion muscle samples were obtained by biopsy of the vastus lateralis with the method of Bergstrom et al. (2). Muscle samples were quickly dissected free of any connective tissue, divided into several pieces, and weighed, with correction made for evaporation of water (11). Muscle samples were stored in liquid N_2 for subsequent analysis of glycogen content. Glycogen concentration of the muscle pieces was determined after they were freeze-dried for ~72 h. The pieces were then weighed and acid hydrolyzed (2 M HCl), and glucose concentration was determined by a standard enzymatic fluorometric assay (23). Blood samples were collected in chilled tubes containing either EDTA or, in the case of samples to be analyzed for lactate, EDTA and sodium flouride. Blood samples were immediately analyzed in triplicate for hematocrit, hemoglobin concentration (Coulter Hemoglobinometer), and plasma protein concentration (American Optical refractometer). Aliquots were centrifuged at 4°C and the plasma separated. Plasma glucose and lactate concentrations were determined using automated analyzers (Yellow Springs Instruments), and the remaining plasma was frozen in liquid N_2 for subsequent analysis of free fatty acid (22) and glycerol (6) concentrations.

Statistical procedures. The data were analyzed using multifactor analysis of variance for repeated measures. Significance of factor main effects and multifactor interactions was determined for the factors trial (i.e., HMG vs. LMG) and immersion (i.e., preimmersion and various times during the immersion). When factor main effects or multifactor interactions were found to be statistically significant ($P < 0.05$), Tukey's critical difference was calculated and used to determine the location of significant differences between means. All data are reported as means ± SE.

RESULTS

Descriptive characteristics of the eight subjects who completed the study are shown in Table 1. Table 1 shows that, although unintentional, the subjects could be separated into two discrete groups on the basis of their body composition. Four of the subjects were relatively lean (<12% body fat) whereas the other subjects were fatter (>17% body fat). Therefore, certain analysis of variance were repeated to determine whether responses differed between the two groups.

The subjects' vastus lateralis glycogen concentrations before and after each of the two cold water immersions are shown in Fig. 1. Preimmersion glycogen concentrations (mmol glucose/kg dry tissue) were lower ($P < 0.001$) for the LMG (144 ± 14) than the HMG (543 ± 53) trial. Postimmersion glycogen concentrations were not significantly different from preimmersion values for either the LMG (141 ± 10) or the HMG (526 ± 53) trials. There were no differences in glycogen concentrations between fat and lean subjects before or after either trial.

There were no significant differences in T_{re} between the LMG and HMG trials either preimmersion (37.10 ± 0.05 and 37.20 ± 0.06°C, respectively) or postimmersion (36.04 ± 0.27 and 36.18 ± 0.26°C, respectively). The individual decreases in T_{re} were calculated and were not significantly different during the HMG trial (1.03 ± 0.21°C) from those during the LMG trial (1.06 ± 0.25°C).

TABLE 1. *Test subject characteristics*

Subject No.	Age, yr	Height, cm	Mass, kg	%Fat	Mean Skinfold, mm	$\dot{V}O_{2\ max}$, ml·kg^{-1}·min^{-1}
1	19	183	73	12	9.9	54
2	20	168	77	21	16.2	53
3	20	188	71	11	7.6	55
4	19	190	102	20	18.4	47
7	20	181	92	22	20.1	54
8	19	173	78	17	15.3	48
9	22	178	64	12	8.2	50
10	21	170	65	12	8.3	45

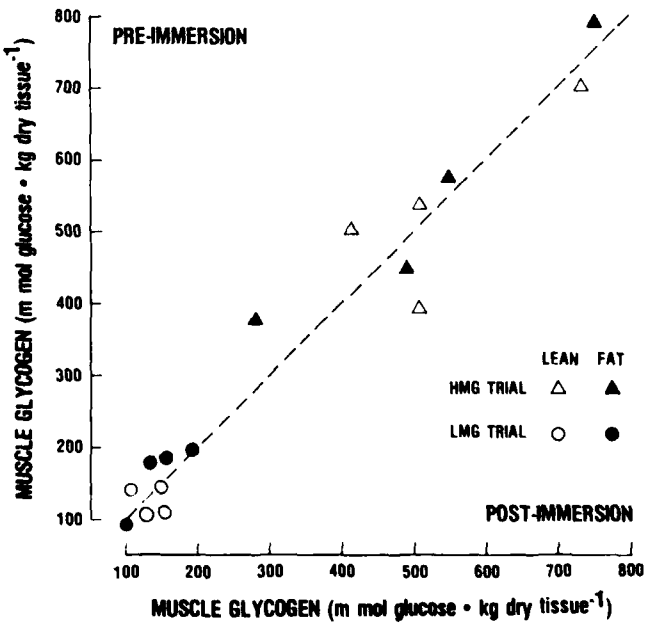

FIG. 1. Comparison of individual pre- and postimmersion vastus lateralis muscle glycogen concentrations during LMG and HMG trials. Dashed line, line of identity.

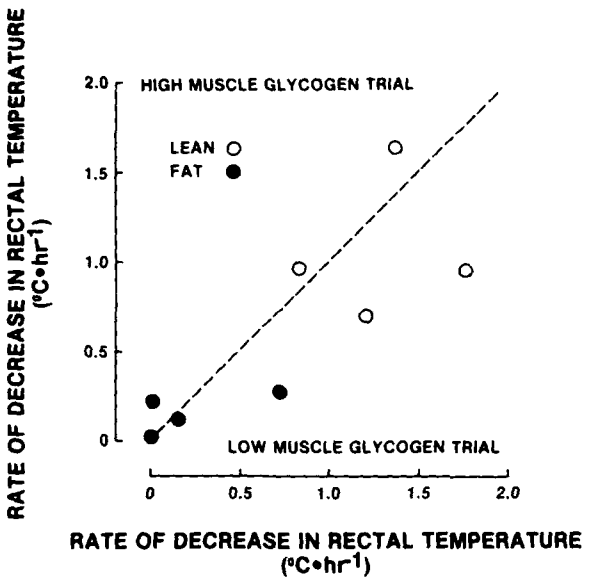

FIG. 2. Comparison of individual rates of decrease in T_{re} during cold water immersion in LMG and HMG trials. Dashed line, line of identity. Lean subjects (○) have ≤12% body fat, and fat subjects (●) have ≥17% body fat.

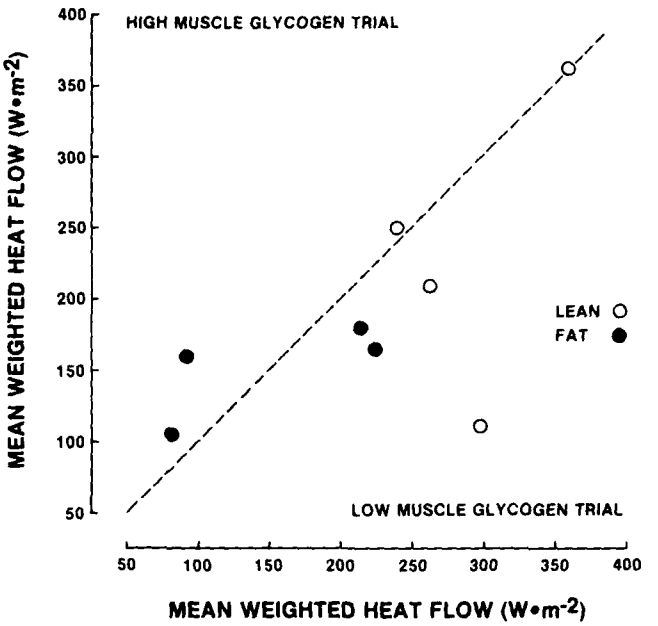

FIG. 3. Comparison of individual weighted skin h_c measurements at the end of cold water immersion in LMG and HMG trials. Dashed line, line of identity. Lean subjects (○) have ≤12% body fat, and fat subjects (●) have ≥17% body fat.

It should be noted that these changes in T_{re} reflect, at least to some extent, the constraints on the subjects' responses imposed by the experimental protocol. For example, several subjects did not complete the full 180 min of immersion during one or both of the trials because their T_{re} decreased below 35.5°C (the predetermined medical safety limit). Furthermore, although there were no significant differences in immersion duration between the LMG trial (123 ± 19 min) and the HMG trial (137 ± 49 min), several subjects achieved the maximum immersion duration (180 min) during both trials, and the experiments were terminated without inducing a substantial fall in T_{re}. Therefore, to facilitate comparisons of the thermoregulatory responses between individuals and trials, the change in an individual's T_{re} was divided by the duration of the immersion. These data are depicted in Fig. 2 (this variable was calculated in this manner and is referred to as a rate of change for simplicity, but it should not be inferred that T_{re} decreased linearly with time as the immersion continued). There were no significant differences between LMG and HMG trials in the rate of decrease in T_{re}. In both trials, the rate of decrease in T_{re} was greater ($P < 0.01$) for lean than fat subjects. Similarly, $\bar{h}^a/_c$ during immersion (Fig. 3) did not differ between trials but was greater in lean than fat subjects during both trials.

The aerobic metabolic rate and respiratory parameters measured during the two immersions are shown in Fig. 4. Shivering was observed in all subjects during both cold water immersions. The ventilatory equivalent for $\dot{V}O_2$ was unaffected by cold water immersion and did not differ between trials. This suggests that the subjects were not hyperventilating and that respiratory exchange ratios were indicative of the relative contributions of fat and carbohydrate oxidation to total metabolic heat production. Respiratory exchange ratios were not altered by cold water immersion but were lower ($P < 0.001$) during

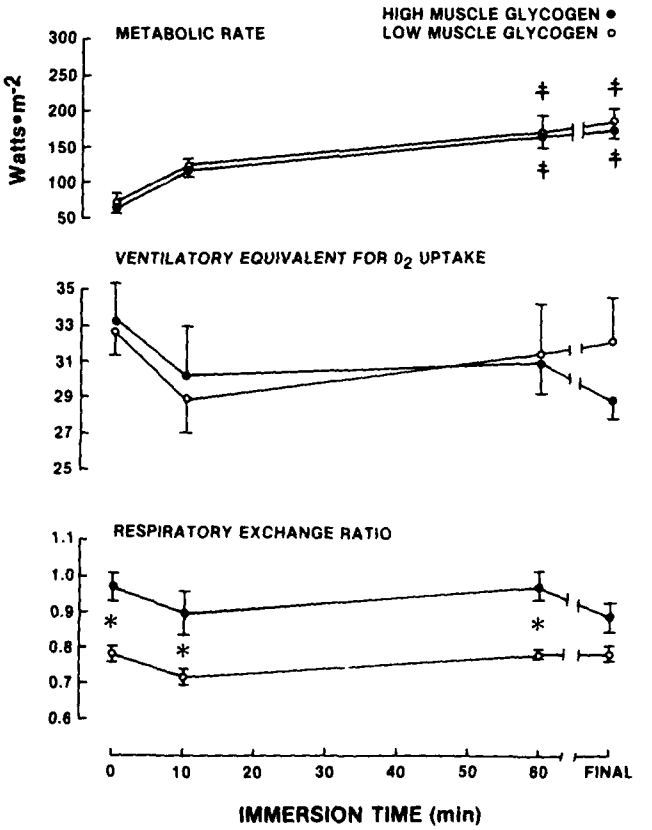

FIG. 4. Metabolic rate, ventilatory equivalent for $\dot{V}O_2$, and respiratory exchange ratio (means ± SE) before (time = 0 min) and during cold water immersion in LMG (open symbols) and HMG (closed symbols) trials. * Significant differences between LMG and HMG trials. ‡ Significant differences from preimmersion value.

the LMG than HMG trial. Metabolic heat production increased with time during cold water immersion, and there were no differences in metabolic rate between the HMG and LMG trials. Initially, there were no differences in metabolic rate between subjects, but, as shown in Fig. 5, the lean subjects exhibited higher ($P < 0.01$) metabolic rates than the fat subjects after 60 min of immersion.

Changes in hematological parameters during the two cold water immersions are shown in Table 2. There were no differences between HMG and LMG for pre- or postimmersion hematocrits, plasma protein concentration, or hemoglobin concentration. Concentrations of hemoglobin and plasma proteins as well as hematocrit increased ($P < 0.001$) during both immersions. These changes correspond to ~8% reduction in plasma volume. However, the accuracy of estimates of plasma volume changes from changes in hematocrit and hemoglobin concentration during cold exposure (28) and water immersion (17) is open to question. For that reason, and because the magnitude of the hemoconcentration was similar and relatively small in both trials, changes in plasma metabolite concentrations were not corrected for plasma volume changes.

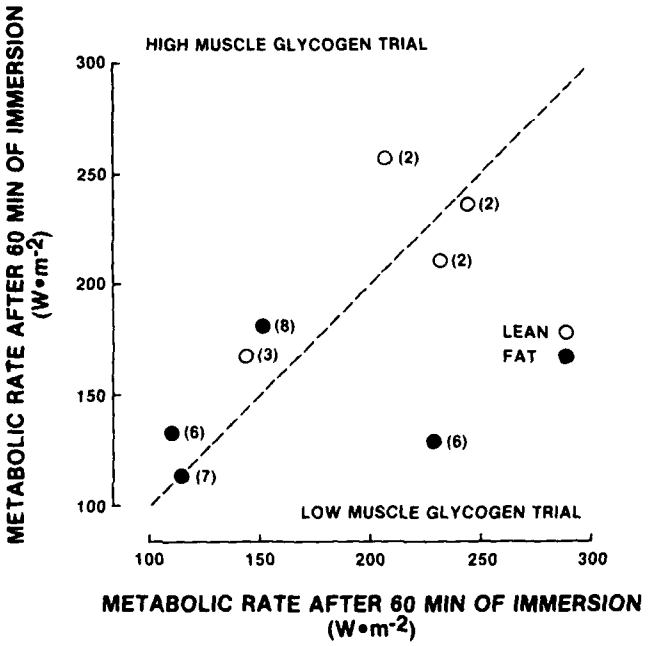

FIG. 5. Comparison of individual metabolic rates after 60 min of cold water immersion in LMG and HMG trials. Dashed line, line of identity. Lean subjects (○) have ≤12% body fat, and fat subjects (●) have ≥17% body fat. Value in parentheses beside each symbol is mean thickness of subcutaneous fat for the subject.

TABLE 2. *Changes in hematological parameters during cold water immersion*

	Preimmersion	Postimmersion
Hematocrit, %		
HMG trial	43.2±0.8	47.6±1.1*
LMG trial	43.4±0.9	48.0±0.7*
Hemoglobin, g/100 ml		
HMG trial	15.0±0.3	16.3±0.3*
LMG trial	15.3±0.2	16.6±0.2*
Plasma protein, g/100 ml		
HMG trial	7.8±0.1	8.6±0.2*
LMG trial	7.5±0.1	8.5±0.1*

Values are means ± SE of measurements made on venous blood samples obtained from subjects who had been resting in a semirecumbent position for >30 min; $n = 8$. There were no significant differences between HMG and LMG muscle glycogen trials for any parameter.
* Significant difference between pre- and postimmersion ($P < 0.001$).

The concentrations of selected plasma metabolites before and during the two immersions are shown in Fig. 6. There were no significant differences in the metabolite concentrations between the lean and fat subjects. Plasma glucose concentrations were higher ($P < 0.005$) both preimmersion and at all times during immersion in the HMG than LMG trial. During both HMG and LMG, there were small but statistically significant ($P < 0.05$) increases in plasma glucose concentration from pre- to postimmersion. Lactate concentrations did not differ between HMG and LMG trials except at the 10th min of immersion when lactates in the HMG trial were higher ($P < 0.005$) than in the LMG trial. Plasma lactate concentration increased ($P < 0.001$) by the 60th min of both cold water immersions compared with preimmersion values. Plasma glycerol concentration increased ($P < 0.001$) during immersion in the LMG trial, and glycerol levels were higher ($P < 0.005$) than in the HMG trial when glycerol levels remained unchanged during immersion. Plasma free fatty acid concentrations were higher ($P < 0.01$) before and during immersion in the LMG trial than in the HMG trial. In both trials, free fatty acids were significantly higher at the end of the immersion compared with preimmersion levels, but the increase was apparent earlier during the LMG than HMG immersion.

DISCUSSION

The diet and exercise manipulation employed in this investigation was successful in achieving the desired preimmersion muscle glycogen states. Preimmersion muscle glycogen levels were substantially different for the two trials. For the low-glycogen trial, preimmersion glycogen concentrations were comparable with those usually observed immediately after 2–3 h of heavy (~70% $\dot{V}O_{2max}$) exercise (2, 9). For the high-glycogen trial, preimmersion glycogen levels were similar or higher than usually observed in subjects consuming an ad libitum mixed diet (2, 9). In addition, although somewhat lower in the low- than high-glycogen trial, plasma glucose levels were in the normal euglycemic range (13) during both trials. Only the vastus lateralis glycogen content was assessed, and undoubtedly the absolute value for glycogen concentration in other muscles differed from that measured in the vastus lateralis. However, it seems reasonable to assume that the effects of the diet and activity regimen were, at least qualitatively, the same for the other muscle groups of the legs, arms, shoulders, and back.

It had been hypothesized that depletion of glycogen stores in several major skeletal muscle groups would impair thermoregulation by limiting shivering during cold water immersion. However, changes in T_{re} during cold water immersion were the same when immersion was preceded by a 3-day program of heavy exercise and low-carbohydrate diet as when the immersion was pre-

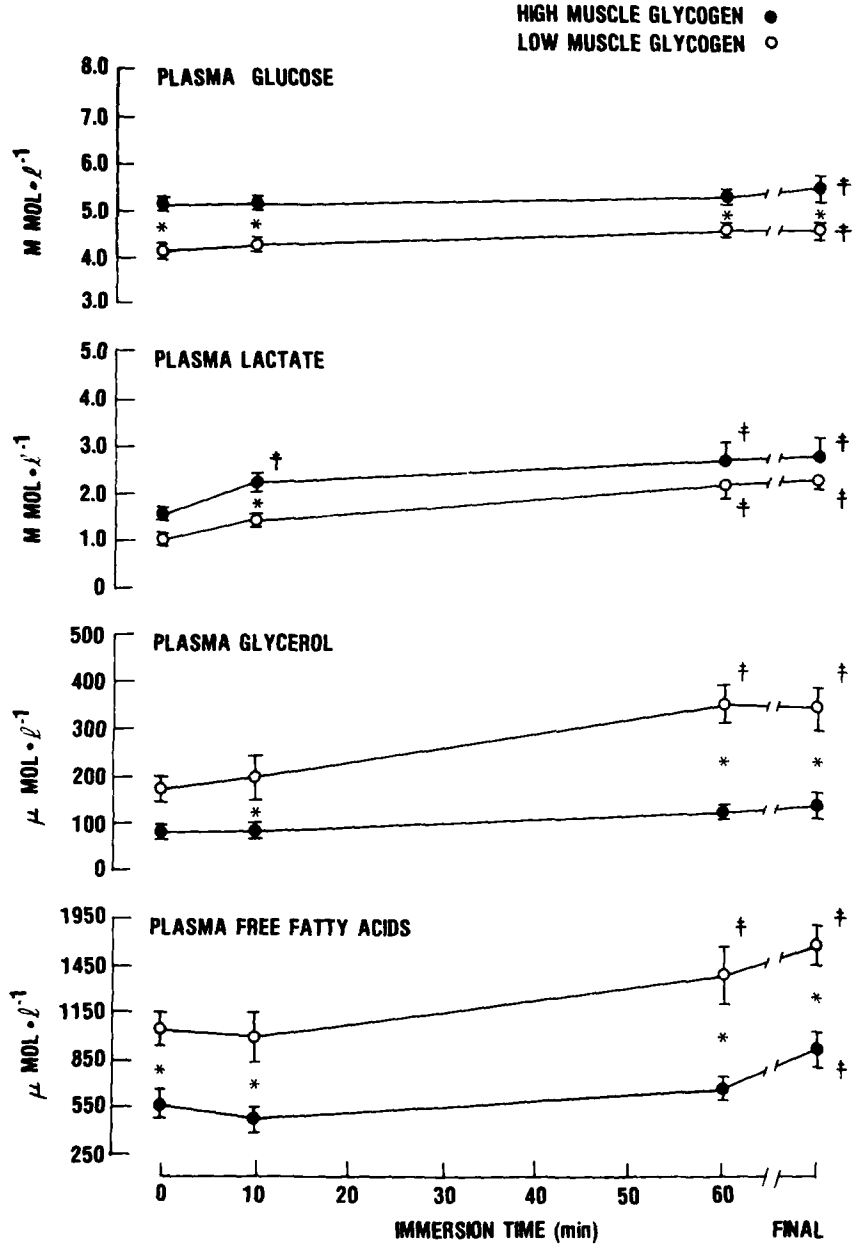

FIG. 6. Plasma metabolite values (means ± SE) before (time = 0 min) and during cold water immersion in LMG (○) and HMG (●) trials. * Significant difference between LMG and HMG trials. ‡ Significant differences from preimmersion value.

ceded by a 3-day program of rest and high-carbohydrate diet. Furthermore, metabolic heat production (Figs. 4 and 5) and skin h_c (Fig. 3) did not differ between the two trials. Thus, the magnitude of whole body shivering and the insulative value of the body's shell during cold water immersion appears to have been unaffected by the preimmersion muscle glycogen concentration.

The basis for the hypothesized impairment of thermoregulation was the assumption that muscle glycogen was the primary metabolic substrate for shivering thermogenesis. There have been studies reported that support this assumption. For example, LeBlanc and Labrie (19) observed an increase in cold tolerance of mice 24 h after completion of an adaptation program consisting of repeated, short-duration exposures to intense (−15°C) cold air. The authors suggested that the improved cold tolerance was associated with the concomitant increase in liver and skeletal muscle glycogen concentration also observed. In contrast, the results of the present investigation indicate that human cold tolerance is not affected by major alterations in skeletal muscle glycogen levels. However, improved cold tolerance of cold-adapted rats and mice is primarily attributed to enhanced nonshivering thermogenesis and diminished shivering (19). Therefore, even if increased liver and muscle glycogen levels do account for improved cold tolerance in mice, shivering metabolism is not likely to be involved.

A greater depletion of vastus lateralis glycogen was observed by Jacobs et al. (16) when subjects exercised in cold air compared with temperate conditions. These investigators concluded that the additional glycogen utilization in the cold was the result of the occurrence of shivering in addition to exercise, although the metabolic

rate measured was about the same in both environments. In the present investigation, no significant changes in vastus lateralis glycogen concentration were observed during either of the two cold water immersion trials in the present investigation. The cold water immersion elicited vigorous shivering, as indicated by the $\dot{V}O_2$ that increased to about the same level (1 l/min) as that of the exercising subjects in the study of Jacobs et al. (16). However, the subjects studied during cold water immersion were, other than shivering, resting quietly. Therefore, shivering does not necessarily result in vastus lateralis glycogen depletion, and mechanisms other than shivering could account for greater glycogen utilization observed during exercise in the cold. For example, Blomstrand et al. (3, 4) have observed that muscle glycolytic flux and ATP utilization during exercise are accelerated by precooling muscle and they suggested that muscle blood flow and/or mechanical efficiency were decreased by cooling the muscle.

The fact that shivering was not associated with a significant muscle glycogen depletion in the subjects of this investigation should not be taken to mean that muscle glycogen could not serve as a substrate for shivering metabolism. For one thing, it is not known whether or not the vastus lateralis was participating in the shivering response during the cold water immersions. Others (12) using electromyographic recordings have documented that cold exposure can elicit shivering of the human quadriceps muscle, but that type of data is not available for the present investigation. On the other hand, observations made in the present study do suggest that there are ample alternatives to muscle glycogen to serve as substrates for shivering thermogenesis either directly via glycolysis or indirectly by enabling glycogen resynthesis at a rate equal to glycogenolysis. Plasma glucose concentrations (and therefore, presumably, hepatic glycogen levels) were within normal levels during both cold water immersions, and insulin-dependent glucose uptake by skeletal muscle has been shown to be enhanced by cold exposure (25). Alternatively, the increase in plasma glycerol and free fatty acids during the low-glycogen immersion trial indicates that enhanced lipolysis could allow muscle glycogen to be spared during shivering metabolism.

Assuming that metabolic control during shivering and exercise are analogous processes, the key determinant of whether shivering results in significant muscle glycogen depletion is most likely the intensity of shivering as reflected by the metabolic rate. It is well known that during exercise, the ratio of carbohydrate to fat oxidation increases as exercise intensity increases. The increase in metabolic rate because of the shivering experienced by the subjects in this investigation was relatively small, compared with moderate-intensity exercise. $\dot{V}O_2$ during shivering only represented ~25–30% of their $\dot{V}O_{2max}$; therefore, it is not surprising that no glycogen depletion was observed. In this regard, the amount of subcutaneous body fat probably plays a role. The lean subjects had higher metabolic rates during immersion than did the fatter subjects (Fig. 5). If even leaner subjects had been studied, a more pronounced increase in metabolic rate and a measurable depletion of muscle glycogen might have been observed. Recently, subjects having ~9% body fat were reported to have experienced a decrease in muscle glycogen as a result of shivering (20).

It has been observed (8) that, regardless of whether other substrates are available, there is some minimal muscle glycogen level that appears to be obligatory to sustain muscle energy metabolism during exercise. Whether this is also true for shivering metabolism is an interesting and important question not yet answered. However, the fact that shivering was unimpaired during the low-glycogen trial, demonstrates that, at least for moderately lean and fat persons, any such critical muscle glycogen concentration necessary to sustain shivering thermogenesis must be very low.

In summary, this investigation has shown that metabolic heat production and body cooling rate of moderately lean and fat individuals immersed in cold water were not altered by a substantial reduction in the muscle glycogen levels of several major skeletal muscle groups. A combination of factors relating to the intensity of shivering and the availability of alternative metabolic substrates probably determine the degree to which muscle glycogen stores are depleted during shivering.

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The views, opinions, and/or findings in this report are those of the authors and should not be construed as official Department of the Army position, policy, or decision unless so designated by other official documentation. Human subjects participated in these studies after giving their free and informed consent. Investigators adhered to AR-70-25 and USMRDC Regulation 70-25 on Use of Volunteers in Research.

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